Visual evoked potentials in rubber factory workers

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Pattern reversal visual evoked potentials (pVEP) were studied in 39 male rubber factory workers in the age range of 18–55 years and 20 control subjects (aged 18–46 years) not exposed to the rubber factory environment. Results revealed that 20 (51%) rubber factory workers had abnormal latencies of wave P1 (dominant component of pVEP) as per accepted criteria of 99% tolerance limit set for the control group (i.e. any value above mean + 3 SD of control was considered abnormal). The section-wise per cent distribution of abnormalities was vulcanization (83%), tubing (75%), calendering (60%), loading (38%) and mixing (14%). This study provides electrophysiological evidence that rubber factory environments affect the conduction processes in optical pathways from their origin in the retina to striate cortex. However, this study has its limitations in not identifying the specific chemical(s) causing these changes in VEP.

Key words: Pattern reversal; rubber factory workers; visual evoked potentials.

INTRODUCTION

The production of rubber articles such as tyres and tubes involves a plethora of complex chemicals. These are grouped into various functional categories such as elastomers, fillers, anti-degradants, solvents, vulcanizing agents, accelerators, activators, retarders, reinforcing agents, anti-tack agents and miscellaneous chemicals. Accordingly, rubber factory workers are exposed to these chemicals which may adversely affect their health and brain functions in particular.

The human nervous system is a highly evolved apparatus for apprehending and perceiving the environment and is the most liable of the body's systems to be damaged by environmental toxins. Hence, many studies have been conducted to assess neurobehavioral effects of occupational exposure to chemicals in industrial workers. Due to subjective errors, the psychometric studies do not confirm the existence of psycho-organic syndrome in industrial workers. Hence, the use of electrophysiological methods such as brainstem auditory, somatosensory and visual evoked potentials can objectively evaluate the toxic effects of chemicals on the central nervous system.

The VEPs assess the functional integrity of the visual pathways from the receptors in the retina to striatal cortex. Any impairment in this pathway will affect VEPs. Most of the neurological disorders of the visual pathway do alter VEPs such as optic neuritis, ischaemic optic neuropathy, toxic amblyopia, dominant optic atrophy, Leber's optic atrophy, optic nerve hypoplasia, glaucoma and tumours. Therefore, the toxic substances which disrupt the pathway will also alter VEPs though the vision can be affected by their toxicity on refractive media of the eye.

The VEPs have been used by various workers to study the neurotoxic effect of organic solvents and metals such as lead and mercury.

The main objective of the present study was to detect changes in VEPs, if any, in rubber factory workers in order to point out possible subclinical impairments of sensory functions and correlate these with the workplace environment.

MATERIALS AND METHODS

The study population comprised 39 male rubber factory workers aged 18–55 years (average age = 27.33 years) engaged in the manufacture of tyres and tubes. They were grouped into various sections of factory work according to their postings, viz. mixing (Section 1), calendering (Section 2), vulcanizing (Section 3), tubing (Section 4) and storage and loading (Section 5). Table 1 shows the section-wise mean age of workers and duration of exposure. Twenty healthy male subjects...
in the age range of 18–46 years served as the control group. They were drawn from the office staff of a medical college, i.e. a population of individuals not exposed to a rubber factory environment or chemical(s) used in a rubber factory. The subjects were brought to the neurophysiology laboratory for VEP study. Their visual acuity was tested and corrected to 6/6. Pattern reversal visual evoked potentials (pVEP) were determined using Neuropack Machine (Nihon Kohden, Japan). Each subject was seated comfortably on a resting chair in front of a black and white TV monitor in a sound- and light-attenuated air conditioned room. pVEPs were recorded from 01 and 02 (international 10—20 notation) referenced to Al and A2 respectively with Fpz as ground.

The pattern test stimulus on the TV monitor was produced by MEB 5200 (Nihon Kohden) evoked potential recorder having an in-built visual pattern generator. The test stimulus was black and white checks with sides 1.4 cm long, subtending an angle of 32' of an arc at a distance of one meter. The pattern reversal rate was 1/sec. The signals recorded were filtered through band spread 1–100 Hz.

The methods used for recording pVEPs were similar to those reported in earlier studies.17-19 Two sets of 256 responses were averaged for each eye and these were analyzed by an inline computer having automatic artefact rejection mechanism. The latencies of various negative and positive waves of evoked potential responses along with amplitude of P1 were calculated. In each subject the average values of latencies of Ni, Pi, and N2, and amplitude of P1 were obtained by adding the values of the right and left eye and dividing by two. VEP abnormalities in the rubber factory workers were identified. The criteria of abnormality were: (1) absence of wave component P1; (2) average value of P1 latency of the exposed case falling beyond mean + 3 SD and (3) an interocular Pi latency difference of 10 m sec or more.20

The mean values of pVEP latencies and amplitudes of exposed and control groups were compared using unpaired student t-test. However, mean values of latencies of wave Ni, Pi and N2 of the exposed group were calculated after excluding the abnormal values (i.e. those values falling beyond mean + 3 SD of the control).

RESULTS

The section-wise distribution of incidence of abnormality in waves N1, P1 and N2 among the different sections of rubber factory workers is shown in Table 2. The percentage of workers showing abnormal latency of wave N1 was (in order of magnitude): Vulcanizing (83%), tubing (62%), calendering (40%) and loading (38%). A similar pattern prevailed for workers with abnormal P1 latency: vulcanizing (83%), tubing (75%), calendering (60%), loading (38%) and mixing (14%). The workers having abnormal latency of wave N2 showed a slightly different trend: tubing (75%), followed by vulcanizing (67%), calendering (60%), mixing (43%) and loading (38%).

Table 3 provides information about a comparison of mean values of latencies of wave Ni, P1 and N2 of all the workers irrespective of sections (excluding abnormal ones) with the control group. The differences between exposed and control group were found to be statistically non-significant. The values of latencies of wave P2 and amplitude of wave P1 of the exposed group without excluding abnormal values when compared with the control group were found to be statistically highly significant and non-significant respectively.

DISCUSSION

This study was conducted specifically to detect sub-clinical involvement of visual pathways as evidenced by neurophysiological changes in workers exposed to a rubber factory environment in comparison with those not exposed. Results revealed that out of 39 workers, 20 (51%) workers had abnormal latencies of wave P1 (the dominant component of VEP) as per accepted criteria of 99% tolerance limit established for the control (Table 2).

These abnormalities may perhaps be due to the exposure of these workers to mixtures of solvents and rubber fumes generated during various processes in the manufacture of rubber products. Major constituents of solvents required for manufacture of tyres and tubes include naphtha, pentane, hexane, heptane, octane, benzene, toluene, xylene and isopropanol and
have been detected in vapours in the ambient environment (air concentration) of selected work areas. These solvents, because of their high affinity for lipids-rich tissue of the brain areas such as brainstem, midbrain and cerebellum, could account for the major changes in latencies along with other chemicals used in the manufacture of the rubber products or formed as a result of chemical reactions during the processes. Many authors have reported prolongation of VEP latencies, particularly P1, in workers exposed to individual solvents such as hexane, toluene and tetrachloroethylene. These studies support the projected view that solvents could be implicated in VEPs abnormalities in rubber factory workers.

The highest prevalence (83%) of abnormalities was found in the workers of the vulcanization section — the section with the least duration of exposure (Table 1). This may be due to more hazardous conditions in this section as during vulcanization solvents such as naphtha, hexane, heptane, isopropanol and toluene used in the lubricants evaporate and contribute to worker's exposure.

High temperatures of 100–200°C further enhance evaporation and increase the ambient concentration of solvents in this process. In the tubing section, spraying of solvents resulted in the exposure of workers. The second highest number of abnormalities (75%) in these workers may be due to longest duration of exposure. The calendering section where 60% of abnormalities have been seen, also employs solvents. The lowest percentage of abnormalities in the mixing section may be due to the fact that this section is rich in the particulate matter which might be responsible for more respiratory problems than those of the central nervous system (CNS) in rubber factory workers. Future studies using detailed qualitative analysis of the workplace environment and its correlation with observed abnormalities in VEPs may pinpoint the chemical/solvent responsible for changes.

However, this study provides electrophysiological evidence that the rubber factory environment delays the conduction processes in optical pathways from their origin in the retina to termination in visual cortex. It is difficult to pinpoint the site where these industrial chemicals affect the visual system as significant changes not only in P1 latency, but also P2, have been observed whose generators might be located higher up in the cortex.

## References


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